to continue, but probably will increase and be buttressed by actions from the Antitrust Division of the Department of Justice.

So once again it appears that medicine and health become the crucible in which social policy for the future is formed. It is easy enough to dismiss all of this simply as harassment, as one more undeserved and malicious attack upon medicine, or as a further expression of antiprofessionalism and anti-intellectualism in our egalitarian society—or even as a federal agency simply doing what it was created to do. But it is more than these. It is yet another rather simplistic approach to solving the problem of the rising cost of health care, and another one based on a false assumption—the assumption this time apparently being that the cost can be controlled if health care is considered as a simple trade and if the rules of the marketplace are imposed upon it. This assumption is apparently made in the face of a substantial amount of evidence that the rules of the marketplace are inappropriate and ill-fitted to the problems of health care, and simply do not work in this area. But beyond this, the FTC approach to health will have profound technologic and social consequences if it is pursued to its conclusion. If the apparatus by which the professions perform their special role in their special disciplines is destroyed, the professions themselves will become impotent and unable to meet the needs for special knowledge and skills which are utterly essential for the smooth working of an increasingly complex technological society. So far as is known there has been no thought given to this outcome, nor has there been any discussion of it. In medicine the FTC approach would return quality control of medical education and patient care to the marketplace, which is precisely where it was before 1910, when there were no professional standards or controls on medical education or patient care.

One can only conclude that this is likely to be a significantly retrogressive step in the further evolution of American medicine and of the American dream. But whether retrogressive or not, it is even more disturbing that a step which is likely to have such profound societal consequences should have been decided upon in the relative secrecy of an independent federal agency with no apparent consultation or discussion with anyone concerning the possible consequences of its actions. But be this as it may, what then is the recourse? The dollar costs of opposing actions of the FTC can be very great and it is already clear that they are beyond the means of many professional organizations who consequently have had no alternative but to capitulate. Perhaps the FTC staff and the commissioners themselves can become better informed of the likely social consequences of dismantling the professions of this nation in today's complex technological world. And perhaps, best of all, this important policy issue can and should be taken to Congress where it should probably have been decided in the first place. It is not yet too late and we urge that this be done soon. This is a civic responsibility in the interest of the citizens of the nation.

—MSMW

Infectious Mononucleosis

Self-Limited Lymphoproliferation

MONONUCLEOSIS SYNDROMES comprise a multitude of diseases characterized by the proliferation of abnormal mononuclear cells recognized in the blood. Three common examples, discussed elsewhere in this issue, are: infectious mononucleosis (IM), cytomegalovirus (CMV) mononucleosis and toxoplasmosis. Also included in this group are a number of other illnesses—rubella, adenovirus infection, infectious hepatitis and many other viral disorders. However, of all these diseases infectious mononucleosis has received the most intense clinical and laboratory investigation during the past ten years. These efforts have not been without success, in fact, the Epstein-Barr virus (EBV) has been identified as the likely cause of IM, the disease which Dameshek first called "a self-limiting leukemia."[1]

In the outstanding review in the Medical Progress section of this issue Fiala and co-workers have exhaustively compared and contrasted classical infectious mononucleosis with the related diseases, cytomegalovirus mononucleosis and toxoplasmosis. Although these three disorders have many common clinical features the authors describe characteristic differences in their clinical courses, epidemiologies and, particularly, laboratory findings. Of these three, only IM is associated with heterophil antibody as well as a large array of other anti-

EBV tests which are available for diagnosis of occasional patients with puzzling manifestations. Actually, CMV mononucleosis and toxoplasmosis are usually readily differentiated from heterophil positive IM by appropriate clinical, microbial and immunologic observations.

The genesis of heterophil antibody, a hallmark of IM, remains one of the interesting unresolved puzzles in this common disease. Heterophil antibodies were first described by Forssman[2] in 1911. Paul and Bunnell[3] subsequently noted their presence in four persons with IM, a disease which had been clearly differentiated from its British cousin, glandular fever, by Sprunt and Evans in 1920.[4] Davidsohn and Walker's[5] landmark discovery that the heterophil antibody of IM differed from the Forssman antibody of serum sickness provided the cornerstone of clinical laboratory diagnosis of IM. That the hetrophil antigen itself is not the cause of IM was shown by Leikola and Aho, who observed typical heterophil antibody responses in the absence of infectious mononucleosis (IM) in a group of volunteers immunized with cells containing the heterophil antigen.[6] Even though heterophil antibody may be an epiphenomenon in IM, its presence is a highly specific and sensitive finding. Fiala and co-workers have correctly pointed out the very low incidence of false negative and positive results for this test.

Clearly, the most exciting recent discovery in the disease is its relationship to the Epstein-Barr virus. Paradoxically, this probable viral cause of IM has provoked new questions regarding EBV's role as a human tumor virus. The discovery that EBV was causally related to IM was made serendipitously in 1967 by the Henles,[7] who were able to establish a continuous lymphoid cell line from the blood of a person only after she contacted IM. Since then virtually all long-term human lymphoblastoid cell lines have been shown to be infected with EBV. Furthermore, the considerable body of seroepidemiologic and virologic evidence compiled in the last ten years leaves little doubt that IM is the result of EBV infection.[8]

Perhaps the most intriguing mystery of EBV's relationship to IM is why the disease is, in fact, self-limiting. Under different circumstances, EBV infections have been causally linked to nasopharyngeal carcinoma and Burkitt lymphoma. A number of possible explanations of this spectrum of responses to EBV infection comes to mind. First, genetic differences in the immune response to many antigens are now recognized and the association of transplantation antigens and disease is well documented. However, no clear-cut genetic predisposition correlating EBV with its associated diseases has been shown to exist. Second, EBV infects B-lymphocytes and in IM there is an intense concomitant proliferation of T-cells presumably in response to the infected B-lymphocytes. Failure of the T-cell population to effectively control the infection might lead to unrestricted B-cell proliferation and eventually a tumor. Finally, socioeconomic, geographical and many other environmental factors are intimately involved in the outcome of EBV infections, especially in view of the high correlation of African Burkitt lymphoma, EBV and endemic malaria. In my opinion, the definitive proof of the existence of human tumor virus may emerge from unraveling the mystery of the subtle host-virus interplay responsible for limited lymphoproliferation in IM.

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REFERENCES

1. Dameshek W, Gunz F: Leukemia. London, Grune & Stratton, Inc. 1964
2. Forssman J: Die Herstellung Hochwertiger Spezifischer Schafhaemolysine ohne Verwendung von Schafblut. Biochem Ztschr 37:78, 1911
3. Paul JR, Bunnell WW: The presence of heterophil antibodies in infectious mononucleosis. Am J Med Sci 183:90, 1932
4. Sprunt TP, Evans FA: Mononuclear leukocytes reaction to acute infection (infectious mononucleosis). Bull Johns Hopkins Hosp 31:410, 1920
5. Davidsohn I, Walker PA: The nature of the heterophil antibodies in infectious mononucleosis. Am J Clin Pathol 5:455, 1935
6. Leikola J, Aho K: Experimentally induced mononucleosis-like heterophil antibodies in man. Clin Exp Immunol 5:67, 1969
7. Henle G, Henle W, Diehl V: Relationship of Burkitt's tumor associated herpes-type virus to IM. Proc Nat Acad Sci USA 39:94, 1968
8. Stites DP, Leikola J: Infectious mononucleosis. Semin Hematol 8:243, 1971

Management of Patients with Aortic Valve Disease

FOR PROPER MANAGEMENT of patients with aortic valve disease, physicians should have knowledge of current information concerning the structural and functional changes that occur in the heart as a consequence of the pressure and volume overload states from aortic valve disease, the relationship of these changes to clinical findings and to prognosis, and the effects of surgical therapy for aortic valve disease. For a number of years, there have been methods available for evaluating the severity of the mechanical defects of aortic valve stenosis and insufficiency.[1,2] More